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Search History

DATE: Tuesday, August 12, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L41	l39 and l4	9	L41
L40	L39 same l4	0	L40
L39	l5 with l11	94	L39
L38	l1 with gene therapy	23	L38
L37	l3 same l8	23	L37
L36	L35 and l5	48	L36
L35	l34 and l15	182	L35
L34	l3 and l20	279	L34
L33	l1 with l2	397	L33
L32	l1 with l4	0	L32
L31	l1 with l20	47	L31

L30	l1 with l15	14	L30
L29	l1 with (l8 or l4)	24	L29
L28	L27 and l4	5	L28
L27	L24 and l20	142	L27
L26	L25 and l20	3	L26
L25	L24 and l1	3	L25
L24	L23 same l5	144	L24
L23	l8 with (l2 or l15)	6656	L23
L22	L21 and l5	6	L22
L21	L20 with l3	31	L21
L20	tumor or cancer	187448	L20
L19	L18 and l4	5	L19
L18	l16 same l8	55	L18
L17	L16 same (l11 or l4)	3	L17
L16	L15 with l5	1068	L16
L15	p53 or p16 or cdk or antisense or p21	218175	L15
L14	l12 and l3	2	L14
L13	L12 same l3	0	L13
L12	L11 with l5	94	L12
L11	inhal$	57019	L11
L10	L9 and l4	14	L10
L9	L8 with l5	915	L9
L8	lung	80567	L8
L7	l5 same l4	0	L7
L6	L5 with l4	0	L6
L5	chemotherapeutic or methotrexate or vinblastine or ectoposide or cyclophosphamide or doxorubicin or cisplatin or camptothecin	39186	L5
L4	nebulized or aerso$	3273	L4
L3	L2 with l1	397	L3
L2	dna or nucleic or plasmid or gene	295279	L2
L1	PEI or polyethylenimine	10785	L1

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L30: Entry 5 of 14 File: PGPB Jul 12, 2001

DOCUMENT-IDENTIFIER: US 20010007902 A1
TITLE: RNASE L ACTIVATORS AND ANTISENSE OLIGONUCLEOTIDES EFFECTIVE TO TREAT TELOMERASE-EXPRESSING MALIGNANCIES

Detail Description Paragraph (42):
[0076] Pharmaceutical compositions suitable for the practice of the invention include solutions of the activator antisense complex in carriers suitable for parenteral administration, such as physiologic saline, sterile water U.S.P., 5% glucose solution U.S.P. and the like. An increased efficiency of intracellular delivery of activator antisense complex can be obtained by complexing the oligonucleotides with polycationic soluble macromolecule or particulate carriers. Suitable particulate carriers include liposomes comprising polycationic lipids (see review Gao, X., & Huang, L., 1995, Gene Therapy 2, 710-722). Specific suitable lipids are described in U.S. Pat. Nos. 5,171,678 and 5,476,962 to Behr et al.; U.S. Pat. Nos. 5,264,618 and 5,459,127 to Felgner et al.; and in Bucherger et al., 1996, Biochemica 2, 7-10. A suitable soluble polycationic carrier that can be used to deliver the activator antisense complex is polyethylenimine, the use of which is described in Boussif et al., 1995, Proc Natl. Acad. Sci. USA 92, 7297-7301.

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Term: L6 with l5

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Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L7	L6 with l5	62	L7
L6	hours or hrs or day or days	1582264	L6
L5	L4 with l3	2205	L5
L4	transgene or dna or polynucleotide or plasmid or adenovi$ or retrovir$ or gene	281464	L4
L3	L2 or l1	16707	L3
L2	cisplatin or VP-16 or vinblastine or ectoposide or cyclophosphamide or doxorubicin	16329	L2
L1	dna damaging agent or dna damaging drug	714	L1

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L7: Entry 25 of 62 File: USPT Apr 1, 2003

DOCUMENT-IDENTIFIER: US 6541603 B1
TITLE: Genes and genetic elements associated with sensitivity to platinum-based drugs

Detailed Description Text (31):
Three days after the last round of infection with the retroviral cDNA fragment library, HeLa or HT1080/ER cells as described in Example 2 were plated in cisplatin-containing media at a concentration of cisplatin which produced approximately 99.9% cytotoxicity in unmodified cells. Selection with cisplatin was followed by G418 selection after growth for three days without any drug selection, using a protocol for each experiment shown in FIG. 3. Each protocol was followed using non-cDNA fragment-containing pLNCX retrovirus-infected cells as a control. After drug selection, increased survival of library-transduced cell populations, relative to the control cell populations, was apparent by microscopic examination in each of the three experiments.

L7: Entry 24 of 62 File: USPT Apr 8, 2003

DOCUMENT-IDENTIFIER: US 6544771 B1
TITLE: Retroviral gene therapy vectors and therapeutic methods based thereon

Detailed Description Text (20):
Accordingly, the invention also provides a method of treating a hematologic disease characterized by a defective gene in a hematopoietic cell in a patient, involving the steps of isolating allogenic, HLA-identical bone marrow cells from a donor; transducing the donor bone marrow cells with a recombinant retroviral vector of the invention engineered to contain a normal gene corresponding to the defective gene at the vector insertion site; culturing the transduced donor bone marrow cells to generate a suitable population of viable cells; destroying the patient's immune system using any suitable method, such as, for example, by the administration of cyclophosphamide (i.e., 50 mg per kg per day for 4 days), or by total body irradiation alone or in combination with cyclophosphamide or other chemotherapeutic agents well known in the art; and, administering a suitable quantity of transfused donor bone marrow cells (approximately 2-6.times.10.sup.8 transfused donor bone marrow cells per kilogram body weight) to the patient via any appropriate route of administration such as, for example, by intravenous infusion, following destruction of the patients immune system.

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L7: Entry 26 of 62 File: USPT Feb 25, 2003

DOCUMENT-IDENTIFIER: US 6524832 B1
TITLE: DNA damaging agents in combination with tyrosine kinase inhibitors

Detailed Description Text (79):
Treatment Protocols 1) Patients exhibiting neoplastic disease are treated with a protein kinase inhibitor, for example genistein, at a concentration of between 1 and 100 .mu.M, or herbimycin A at a concentration of between about 1 and 100 .mu.M, for 6 hours prior to exposure to a DNA damaging agent. 2) Patients are exposed to ionizing radiation (2 gy/day for up to 35 days), or an approximate a total dosage of 700 gy. 3) As an alternative to ionizing radiation exposure, patients are treated with a single intravenous dose of mitomycin C at a dose of 20 mg/m.sup.2.

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L7: Entry 36 of 62 File: USPT Aug 7, 2001

US-PAT-NO: 6271207
DOCUMENT-IDENTIFIER: US 6271207 B1
**** See image for Certificate of Correction ****

TITLE: Enhanced expression of transgenes

DATE-ISSUED: August 7, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Cristiano; Richard J.	Pearland	TX		
Nguyen; Dao	Potamac	MD		

US-CL-CURRENT: 514/44; 424/93.2, 435/320.1, 435/455, 435/458

CLAIMS:

What is claimed is:

1. A method for enhancing the expression of a transgene in a target neoplastic cell in vivo comprising:

(a) administering a DNA-damaging agent to a subject containing a target neoplastic cell; and

(b) transferring said transgene into said target neoplastic cell between 2-4 days after said administering step;

whereby expression of said transgene is enhanced as a result of the administering of said DNA-damaging agent to said target neoplastic cell.

2. The method of claim 1, wherein said target neoplastic cell is a dividing cell.

3. The method of claim 1, wherein said DNA-damaging agent is selected from the group consisting of cisplatin, carboplatin, VP16, teniposide, daunorubicin, doxorubicin, dactinomycin, mitomycin, plicamycin, bleomycin, procarbazine, nitrosourea, cyclophosphamide, bisulfan, melphalan, chlorambucil, ifosfamide, merchlorehtamine, and ionizing radiation.

4. The method of claim 1, wherein said transgene is transferred at about 3 days after said administering step.

5. The method of claim 1, wherein said transfer of said transgene is accomplished by a technique selected from the group consisting of liposome-mediated transfection, receptormediated internalization and viral infection.

6. The method of claim 1, wherein said transgene encodes a tumor suppressor.

7. The method of claim 6, wherein said tumor suppressor is p53.

8. The method of claim 7, wherein said p53 transgene is under the transcriptional control of a CMV IE promoter.

9. The method of claim 3, wherein said DNA-damaging agent is cisplatin.

10. The method of claim 7, wherein said p53 transgene is carried in an adenoviral vector.

L7: Entry 47 of 62 File: USPT Nov 10, 1998

DOCUMENT-IDENTIFIER: US 5834182 A
TITLE: Method for increasing transduction of cells by adeno-associated virus vectors

Detailed Description Text (66):
The following study demonstrated that episomal vector DNA amplification does not explain increased transduction. Helper virus-independent amplification of wild-type adeno-associated virus DNA has been reported to occur following genotoxic stress (Yalkdinoglu A. O. et al., Cancer Res. 48, 3124-3129 (1988)). More than 400-fold amplification has been observed in CHO-K1 cells following treatment with the mutagen N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) and a 30-fold amplification in human diploid fibroblasts (E6). To determine whether a similar phenomena might accompany the increased transduction efficiency of AAV vectors in cells exposed to DNA damaging agents, Hirt supernatants from both irradiated and unirradiated cultures of stationary primary human fibroblasts were assayed 48 hours following vector exposure. Quadruplicate cultures received either no treatment, vector alone or both 4000 rad of gamma irradiation and vector. At 48 hours low molecular weight DNA was isolated from triplicate cultures in each treatment group. The fourth culture in each group was stained for alkaline phosphatase-positive cells to determine the increase in transduction efficiency caused by the gamma irradiation, which was in excess of 100-fold. An autoradiograph of low molecular weight DNA isolated from triplicate stationary cultures of primary human fibroblasts in each of three treatment groups was made. The groups were control uninfected cultures, unirradiated cultures infected with AAV-LAPSN and cultures infected with AAV-LAPSN after 4000 rad of gamma irradiation. Briefly, Hirt supernatant DNA, which was harvested from the triplicate cultures from each treatment group 48 hours after infection, was subjected to Southern analysis using a neo probe. A phosphoimager was used to quantitate the total hybridization signal in each lane, and the signal representing the single stranded monomer forms of vector DNA. The maximum variation between lanes was 45% i.e., within experimental error. The results revealed no evidence of significant DNA amplification in gamma irradiated cultures. These data demonstrated that the increased transduction efficiency of AAV vectors in irradiated cells was not due to marked amplification of episomal vector DNA.

L7: Entry 48 of 62 File: USPT May 5, 1998

DOCUMENT-IDENTIFIER: US 5747469 A
**** See image for Certificate of Correction ****
TITLE: Methods and compositions comprising DNA damaging agents and p53

CLAIMS:

80. The method of claim 79, wherein the period between administration of the viral vector and DNA damaging agent is between 12 and 24 hours.

81. The method of claim 79, wherein the period between administration of the viral vector and DNA damaging agent is between 6 and 12 hours.

82. The method of claim 79, wherein the period between administration of the viral vector and DNA damaging agent is about 12 hours.

84. The method of claim 83, wherein the period between administration of the DNA damaging agent and viral vector is between 12 and 24 hours.

86. The method of claim 83, wherein the period between administration of the DNA damaging agent and viral vector is about 12 hours.

L7: Entry 61 of 62 File: JPAB Aug 15, 1995

DOCUMENT-IDENTIFIER: JP 07215882 A
TITLE: TREATMENT OF TUMOR

Abstract Text (2):
CONSTITUTION: A cytotoxicity-enhancing dose of a compound of the formula [X is H, a 1-4C hydrocarbyl, a halogen, OH, etc.; (n) is 0, 1; Y1, Y2 are each H, nitro, a halogen, a (substituted) 1-14C hydrocarbyl, etc.] such as 1,2,4- benzotriazine, to a mammal having a solid tumor susceptible to treatment with chemotherapy agents to one to two hours after the administration of a DNA- alkylating chemotherapy agent such as cisplatin or to 1/2 to 24 hours before the administration of the chemotherapy agent to enhance the cytotoxicity of the chemotherapy agents. The benzotriazine oxide is preferably administered at a dose of 30-250 mg/m2.